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NECROTIC ENTEROTOXAEMIA IN A CALF DUE TO *CLOSTRIDIUM WELCHII* TYPE B

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INTRODUCTION

This paper records a case of dysentery and necrotic enterotoxaemia due to *Clostridium welchii* Type B (the lamb dysentery bacillus) in a young calf suckling its dam. This calf was one of five which succumbed during an outbreak of dysentery among calves born on a farm in the spring of 1951.

The occurrence of clostridial infections in calves and adult cattle has rarely been recorded, *e.g.*, only *Cl. chauvoei* infections being referred to in the Report on Diseases of Calves (N.V.M.A., 1947). Bosworth (1943) has demonstrated the existence of a new type of *Cl. welchii* associated with enteritis in young calves. At the Newcastle laboratory *Cl. welchii* Type A has been recovered several times from blackleg lesions in cattle, together with either *Cl. septicum* or *Cl. oedematiens*, while in 1948 *Cl. welchii* Type D was isolated in pure culture from the heart blood of a yearling heifer dead from acute meningitis.

HISTORY OF THE OUTBREAK

An outbreak of dysentery with occasional deaths occurred amongst young calves born between mid-March and mid-May, 1951, on a farm west of Newcastle, this farm lying at about 600 feet above sea level. Grazings are predominantly exposed permanent and temporary pastures and meadows.

A herd of 27 Kyloe (Shorthorn × Highland) cows and 11 Blue Grey (Shorthorn × Galloway) cows is maintained and the policy is to commence calving in late March, the Kyloe cows calving first. The cattle lie out throughout the year and calve outside, the calves remaining with the dams until weaned. One of the earlier calves, when about a week old, showed a severe scour which developed into dysentery. This condition did not respond to sulphonamide and penicillin treatment and the calf died on March 27th. *Post-mortem* examination made on the farm showed a severe generalised enteritis.

Up to April 17th a further 21 calves had been born and 16 of these developed a severe scour, several calves passing a little blood. Calves were usually affected when about 7 to 10 days old. These 16 all received large doses of a stock polyvalent antiscour serum (containing an anti-*Bacterium coli* fraction), were carefully nursed and received repeated oral administration of glucose, but between April 17th and May 8th four of them died, the last casualty, calf 5, being received for *post-mortem* examination at this laboratory. The course of the disease varied but was generally rather rapid ; death occurred overnight in one case while others lasted two, three, four or more days. Recovery in the other affected calves was slow, taking 10 to 14 days in some cases.

On *post-mortem* examination calf 5 showed severe intestinal lesions and initial biological tests carried out, using material present in the intestine, demonstrated an enterotoxaemia due to *Cl. welchii* Type B or C, this being later established as being due to Type B.

Prophylactic treatment with lamb dysentery serum was therefore recommended and all calves born subsequently, together with those born before May 8th, were given injections of 10 ml. concentrated lamb dysentery antiserum, but before this treatment was instituted four recently born calves commenced scouring when three to four days old. These rapidly developed symptoms of toxaemia and dysentery and were regarded as typical cases of the disease under investigation. As soon as this serum was procured 40 ml. were given to each calf and all recovered within two to three days.

Thus, from this herd a total of 37 calves were born of which 21 developed scouring, some with subsequent dysentery, of which five died.

It was found that there was a long-standing history of lamb dysentery amongst lambs on this and other farms in the district, a patent outbreak occurring on this farm in 1950. Routine vaccine prophylaxis was used for the 1951 lambing season, no case of lamb dysentery being reported.

Post-mortem FINDINGS

The most significant macroscopic lesions were located in the small intestine which showed severe generalised enteritis with extensive necrosis of the mucous membrane, particularly in the ileum. Small yellowish diphtheritic patches were distributed irregularly in these necrotic areas. The contents of the small intestine were liquid and blood stained. Slight congestion of the mucous membrane of the caecum and colon was noted, while there was some gelatinous oedema of the mucous membrane lining the abomasum. The mesenteric lymph nodes were very swollen and showed severe haemorrhages. Liver, spleen and kidneys were intensely congested with blood but the lungs appeared normal. A large haemorrhagic patch covered much of the epicardium of the left ventricle but no endocarditis was observed.

Histological examination of the necrosed and inflamed areas of the small intestine showed extensive inflammatory changes. The mucous membrane overlying the villi was necrosed, with occasional complete destruction of villi. Examination of the mesenteric lymph nodes showed an active vascular reaction with hyperaemia of the venulae and extensive cellular infiltrations. When sectioned the kidneys showed subcapsular haemorrhages, swelling of the convoluted tubules, large areas of congestion in the medulla, and cellular degeneration with some nuclear fragmentation. These changes were suggestive of a commencing necrosis.

These lesions, both macroscopic and microscopic, in many respects resembled those seen in typical lamb dysentery of the acute type. Microscopic examination of direct smears from the necrosed areas, stained by Gram's method, showed among other organisms many short, square-ended, gram-positive rods resembling *Cl. welchii*. Smears examined from heart blood, liver and spleen showed no organisms.

BIOLOGICAL TESTS

The contents of the small intestine were centrifuged and the supernatant fluid clarified through Zeitz K5 discs. The resulting filtrate proved highly toxic to mice, 0·3 ml. given intravenously killing in four to five minutes, the mice exhibiting severe nervous spasms before death, while 0·02 ml. killed mice in 20 to 24 hours.

Mixtures of 0·9 ml. filtrate and 0·3 ml. various clostridial antitoxic sera were made, left at room temperature for 30 minutes and 0·4 ml. of the mixture injected intravenously into pairs of mice. The results were :—

Antitoxic serum added	*Effect on mice*
Cl. welchii Type A	Both died< five minutes.
Cl. welchii Type C	Both lived.
Cl. welchii Type D	Both died< five minutes.
Cl. welchii Type B, *i.e.*, C + D (0·5 ml. mixture injected as two antisera used)	Both lived.
Cl. septicum	Both died< five minutes.

Intradermal tests in guinea-pigs using this filtrate showed the presence of a necrotic toxin inhibited by *Cl. welchii* Types B and C antisera but not by *Cl. welchii* Types A or D or *Cl. septicum* antisera. These tests demonstrated the presence of Beta toxin of *Cl. welchii*.

Using the methods of Bosworth and Glover (1934) Epsilon toxin of *Cl. welchii* was demonstrated by activating the filtrate with trypsin, when toxin inhibition tests in mice showed :—

Antitoxic serum added	*Effect on mice*
Cl. welchii Type C	Both died one and a half to two hours.
Cl. welchii Type D	Both died 15 minutes.
Cl. welchii Type B, *i.e.*, C + D ...	Both lived.

Thus the main lethal toxins of *Cl. welchii* Type B were identified (Dalling & Ross, 1938 ; Oakley, 1943) and a diagnosis of necrotic enterotoxaemia due to this organism was given.

Isolation of the Causal Organism

Cultures made on ox blood agar and cooked meat medium from heart blood, liver, spleen, kidney and bone marrow remained sterile after incubation aerobically and anaerobically at 37° C. for four days. Ox blood agar plates, sown directly from ileum contents and necrotic lesions and incubated anaerobically, yielded among other organisms many haemolytic colonies resembling those of *Cl. welchii*. These colonies were generally absent from plates inoculated from abomasum and caecal contents. No evidence of the presence of Salmonella organisms was obtained.

Single colonies of *Cl. welchii* were subcultured into tubes of cooked meat medium, incubated overnight and thence into 60 ml. quantities of this medium. After incubation for five hours the broth was clarified through Zeitz K5 discs. The filtrate was then subjected to detailed examination and the various toxins identified as follows :—

Toxin	*Identification*
Alpha ...	Opalescence in egg yolk extract. Haemolysis of ovine erythrocytes. Inhibited by *Cl. welchii* Type A antiserum.
Beta ...	Toxic to mice and guinea-pigs. Inhibited by *Cl. welchii* Type C antiserum.
Epsilon ...	Toxic to mice after activation with trypsin. Inhibited by *Cl. welchii* Type D antiserum.

On older cultures it was shown that both Beta and Epsilon toxins were present at 48 hours incubation but Epsilon only at 72 hours and later. This strain of *Cl. welchii* was compared throughout these tests with known *Cl. welchii* Types B (CN1990 of B. W. & Co.) and C (CN883 of B. W. & Co.), an exact comparison being obtained with the Type B strain. It was therefore concluded that this was a typical strain of *Cl. welchii* Type B. A subculture of this strain sent to the Wellcome Research Laboratories, Beckenham, was confirmed as *Cl. welchii* Type B.

Discussion

Necrotic enterotoxaemia in lambs due to *Cl. welchii* Type B, although identified as such in 1926, has not been recorded in other animals apart from the report of the disease in foals (Mason & Robinson (1938) in South Africa), and the demonstration by Montgomerie and Rowlands (1937) of *Cl. welchii* Type B in intestinal lesions in a foal suggestive of lamb dysentery.

Although only one calf from this outbreak was examined in this centre, the marked response to lamb dysentery serum appeared to be significant, especially when taken in conjunction with the symptoms, lesions, biological and bacteriological findings. Also the veterinary surgeon noted severe bowel inflammation in a *post-mortem* examination of the first dead calf. It would seem probable, therefore, that some at least of the other deaths were due to this disease.

The environmental conditions existing at the time necrotic enterotoxaemia occurred in these calves compared in many respects with those associated with the development of dysentery in lambs, *i.e.*, the cows calve outside and the calves suckle their dams, but the following difference was noted. Many of the calves affected with scour recovered with treatment including lamb dysentery serum, although it cannot, of course, be assumed that these were all subjects of *Cl. welchii* Type B infection.

With the emphasis on beef-producing herds on marginal and hill farms in Great Britain the possibility of transference of infection from sheep to cattle and cattle to sheep cannot be ignored.

Summary

A case of necrotic enterotoxaemia in a young calf suckling its dam is described and shown to have been due to infection with *Cl. welchii* Type B.

The methods of identification of the various toxins found in the small intestine and the isolation and identification of the causal organism are described. Four other deaths in calves out of a total of 37 calves born in the same herd may have been due to the same disease, and the use of concentrated lamb dysentery serum in injections of 40 ml. cured four affected calves and prevented further scouring and dysentery in the last 11 calves to be born, these being injected soon after birth.

A comparison is made between necrotic enterotoxaemia in calves and dysentery in lambs.

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References

Bosworth, T. J. (1943.) *J. comp. Path.* **53.** 245-255.
——— & Glover, R. E. (1934.) Rep. Inst. Anim. Path., Camb. **4.** 79.
Dalling, T., & Ross, H. E. (1938.) *J. comp. Path.* **51.** 235-249.
Mason, J. H., & Robinson, E. M. (1938.) *Onderstepoort J. vet. Sci.* **11.** 333-337.
Montgomerie, R. F., & Rowlands, W. T. (1937.) *Vet. Rec.* **49.** 398.
N.V.M.A. (1947.) Report of Survey Committee on Diseases of Farm Livestock : Diseases of Calves. N.V.M.A. Publication No. 12.
Oakley, C. L. (1943.) *Bull. Hyg.* **18.** 781-806.

COMPARISON OF THE TOXIC EFFECTS OF FOUR TRYPANOCIDAL SUBSTANCES FOR EAST AFRICAN ZEBU TYPE OF CATTLE

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As the result of work at this laboratory on the toxicity of dimidium bromide (1553) (Burdin & Plowright, 1952) and phenidium chloride (897) (Burdin *et al.*, 1952) in normal Zebu cattle, samples of two more phenanthridinium compounds were made available for study. These were 2:7-diamino-9-thienyl-10-methylphenanthridinium bromide (referred to as No. 621.C.47) and 2:7-diamino-9-p-aminophenyl-10-methylphenanthridinium chloride (referred to as No. 150.C.47). Although they have not been used in the bovine in the field, pilot observations by Goodwin (1951) showed them to have therapeutic activity against trypanosome infection in Nigerian cattle.

Orginally it was intended that the observations recorded here should be on a small number of animals as a preliminary to experiments on larger numbers if the drugs promised to be suitable for field use. The results, however, are fairly conclusive in themselves and no further work on these particular substances is contemplated.

Dimidium bromide was introduced in this trial in an attempt to exclude the effect of seasonal variation in atmospheric and grazing conditions on any toxicity produced, *i.e.*, to act as a positive control. Antrycide was included to observe whether this substance could produce delayed liver damage of a nature similar to that detected after the administration of dimidium bromide, because of certain points of similarity in their chemical structure.

Technique

Twenty-four yearling heifers of Boran Zebu type were used for the experiment; they were grazed with six control animals obtained from the same source, these also serving as controls for the phenidium chloride trials running concurrently (Burdin *et al.*, 1952).

Inoculations with freshly prepared solutions were made on July 10th, 1951, as follows:—

Table I
Protocol of Bovines Used

Bovine Nos.	Treatment
2094 & 2113	1 mg. per kg. dimidium bromide s/c. as 2% solution
2105 „ 2120	3 „ „ „ „ „ „
2095 „ 2114	5 „ „ „ „ „ „
2101 „ 2111	1 „ 621.C.47 „ „ „
2106 „ 2118	3 „ „ „ „ „
2096 „ 2110	5 „ „ „ „ „
2102 „ 2116	1 „ 150.C.47 „ „ „
2097 „ 2108	3 „ „ „ „ „
2103 „ 2109	5 „ „ „ „ „
2100 „ 2104	5 „ antrycide „ 4% „
2099 „ 2112	7·5 „ „ „ „ „
2107 „ 2098	10 „ „ „ „ „
17 „ 18	Controls
20 „ 21	„
24 „ 30	„

All animals were weighed weekly and subsequently bled for biochemical determinations.

Clinical Observations and Bodyweight Records

No symptoms were noted immediately after injection with the phenanthridinium drugs. Within a few minutes of the antrycide inoculations, however, the striking facial twitching, salivation and other parasympathomimetic effects were evident (Garner, 1950).

Dimidium Bromide Groups.—Loss in weight occurred in all animals except No. 2113, which received 1 mg. per kg. It was most marked in those treated with 3 mg. per kg. and these also showed typical skin lesions. No. 2105 at day 63 post inoculation was 100 lb. below starting weight. "Photosensitisation" lesions were first noticed at day 50 p.i. and were almost identical in both heifers, which, incidentally, had black coats. In addition to ear and muzzle lesions previously described (Plowright *et al.*, 1952) the vulva and anus of both animals were severely involved in a process leading to hardening and subsequent sloughing of the exposed epithelium. The mucosa of the tongue as far back as the dorsum was necrotic and this, together with involvement of the inner aspect of the lips and gums, led to a most offensive odour emanating from their mouths, with profuse salivation and difficulty in feeding.

Death of the animals on 5 mg. per kg. was preceded by loss of weight and condition, the only premonitory symptom being epistaxis from one nostril in No. 2095. Both became comatose before death.

621.*C*.47 *Groups.*—Clinical evidence of toxicity was observable only in No. 2096 (dosed with 5 mg. per kg.), which lost weight. In this animal, as will be seen from the pathological report, the periportal liver lesion typical of dimidium toxicity was found.

150.*C*.47 *Groups.*—All animals lost weight except for No. 2097, which died at day 33 p.i. The decrease in weight appears to be more gradual than with dimidium bromide and in Nos. 2108 and 2109 to be continuous from the time of injection of the drug.

Antrycide Groups.—Nos. 2107 and 2098, dosed at 10 mg. per kg., died so rapidly that weight records showed no significant change. They were first noticed to be ill some 12 hours before death, with mucosae pallid, respiration laboured and pulmonary oedema easily detectable on auscultation of the thorax. When forced to walk, a clear frothy exudate escaped from the nostrils and occasionally formed "soap-bubbles" which drifted away in the air. Eventually a bubbling noise on breathing could be heard several yards away and they became comatose and died.

No. 2112, which received 7·5 mg. per kg., lost weight rapidly to a minimum of 110 lb. below starting weight at day 21 p.i. Recovery was very slow and at day 77 p.i. she was still 60 lb. below normal.

No. 2099, also at 7·5 mg. per kg., and both those at 5 mg. per kg. remained quite healthy with no clinical abnormality detected.

Controls.—These remained normal throughout and weights were maintained though not increased.

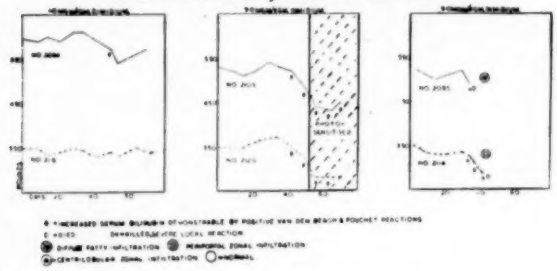
Biochemical Observations

Haemoglobin, serum alkaline phosphatase estimations and van den Bergh and Fouchet reactions were performed as in a previous communication (Burdin & Plowright, 1952).

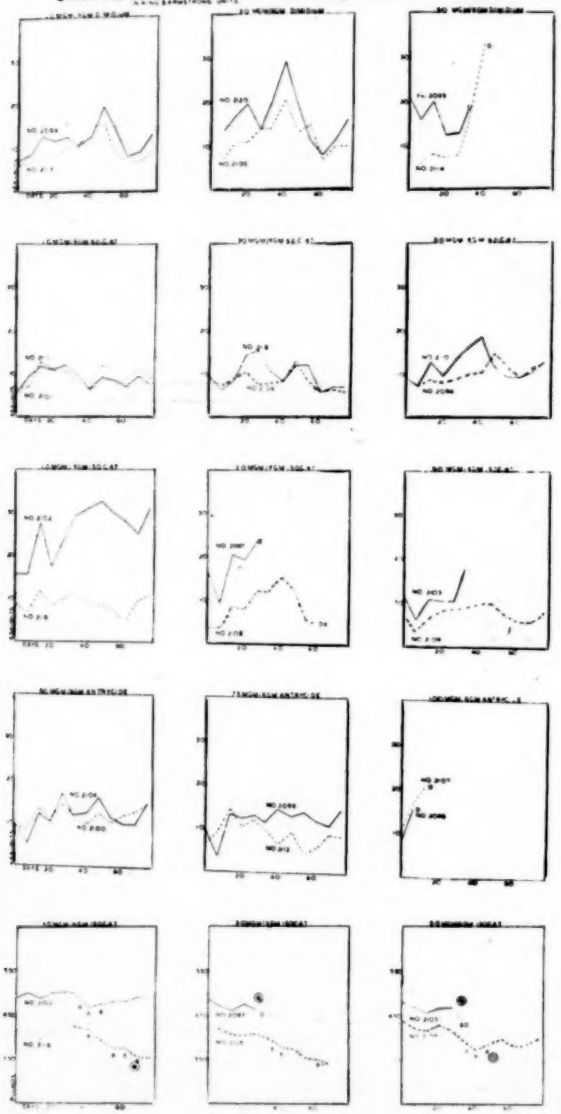
Haemoglobin in all phenanthridinium-treated groups

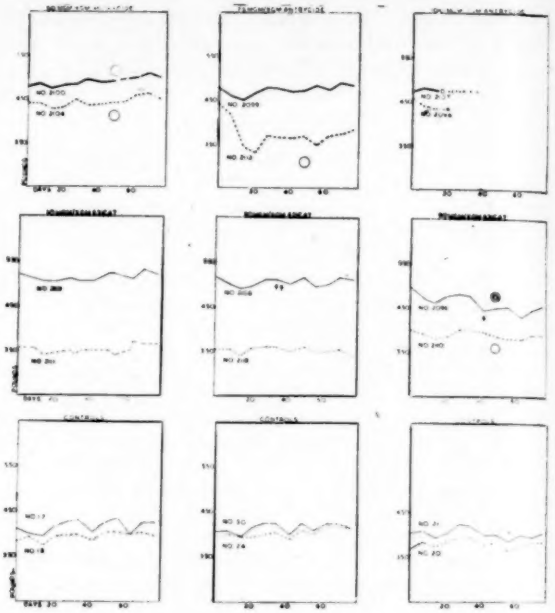
remained normal. Antrycide-treated animals showed variations to be described below. Van den Bergh positive reactions, bodyweights and serum alkaline phosphatase values are recorded in Graphs I and II.

GRAPH I. BODYWEIGHT / TIME POST INOCULATION.



GRAPH II SERUM ALKALINE PHOSPHATASE / TIME POST INOCULATION





Dimidium Bromide Group, 1 *mg. per kg.*—Both animals were van den Bergh positive on one occasion only. They showed maximum phosphatase values at day 49.

Dimidium Bromide Group, 3 *mg. per kg.*—Both heifers were van den Bergh positive from day 42 onwards and No. 2105 remained so until day 70 p.i., No. 2120 becoming negative at day 63 p.i. The peak in the phosphatase curves occurred at day 42 p.i.

Dimidium Bromide Group, 5 *mg. per kg.*—No. 2095 was van den Bergh positive at examination on day 37 p.i. just before death, with no observable change in serum phosphatase content. No. 2114 became van den Bergh positive on day 33 p.i. and remained so until death. Phosphatase values rose to the high figure of 33 units just before death.

621.*C*.47 *Group*, 1 *mg. per kg.*—Van den Bergh negative throughout and no definite changes in phosphatase levels.

621.*C*.47 *Group*, 3 *mg. per kg.*—No. 2106 was van den Bergh positive at days 34 and 37 with a slight rise in phosphatase at day 49. No. 2118 was van den Bergh negative but exhibited a more marked rise in phosphatase, with maximum at day 38.

621.*C*.47 *Group*, 5 *mg. per kg.*—No. 2096 was van den Bergh positive on day 42 only, with a slight rise in phosphatase at day 49. No. 2110 was van den Bergh negative but with a pronounced rise in phosphatase to a maximum at day 42.

150.*C*.47 *Group*, 1 *mg. per kg.*—Both animals became van den Bergh positive, No. 2116 remaining so until day 70, and in one, No. 2102, a marked rise in phosphatase occurred with a peak at day 49.

150.*C*.47 *Group*, 3 *mg. per kg.*—One subject, No. 2097, died without excess bilirubin in the serum but with rapidly rising phosphatase. The other, No. 2108, first became van den Bergh positive at day 37 and remained so until killed *in extremis* at day 63 having maximum phosphatase at day 42.

150.*C*.47 *Group*, 5 *mg. per kg.*—No. 2103 died at day 39 having developed a positive van den Bergh reaction at day 34, associated with elevated phosphatase. No. 2109 was

van den Bergh positive on three occasions, commencing at day 37 p.i. A slight rise in phosphatase occurred.

Antrycide Groups.—On no occasion was the van den Bergh reaction positive in antrycide-treated animals. Phosphatase was not significantly altered except in the two animals at 10 mg. per kg. which died early in the experiment with values elevated above the starting point.

In these experiments the only variations from normal haemoglobin occurred in the antrycide groups where, just before death, the bloods from the 10 mg. per kg. animals became unreadably low. The plasma was water-clear and although haematocrit readings were not taken, the volume of red cells was obviously markedly decreased. A fall in haemoglobin to 7·1 g. per 100 c.c. at day 49 occurred in No. 2112 at 7·5 mg. per kg.

Controls.—Biochemical determinations were only continued until day 28, but up to then they remained normal. Weight records, however, were maintained for the whole period of the experiment. These animals, used as controls for another toxicity trial, had been bled weekly for the previous 47 days and had been normal throughout.

Pathological Observations

The animals from which tissues were obtained are listed in Table II. It can be seen that biopsies were carried out weekly between the 35th and 50th days, or until death, on all animals which received 5 mg. per kg. of the various drugs. The technique employed was that described in previous papers of this series (Plowright & Burdin, 1952; Burdin & Plowright, 1952), and only frozen sections stained for fat were examined. *Post-mortem* material was obtained from various animals as shown in Table II. Frozen sections of liver and kidney were stained for fat, paraffin sections by routine methods.

Table II
Material Obtained for Histological Examination

Animal No.	Days post-inoculation	Tissues
2095	35	Liver biopsy
	41	Liver, lymph gland, adrenal, kidney, abomasum, spleen, salivary gland
2114	35, 42	Liver biopsy
	45	Liver, adrenal, spleen, myocardium, kidney, salivary gland, pancreas
2096	35, 42, 49, 56	Liver biopsy
2110	35, 42, 49, 56	Liver biopsy
2116	77	Liver, kidney
2097	33	Liver, kidney, spleen, adrenal, salivary gland
2108	40	Liver, kidney
2103	35	Liver biopsy
	39	Liver, kidney, myocardium, muscle at site of inoculation, abomasum
2109	35, 42, 49, 56	Liver biopsies
2100	35, 42, 49, 56	Liver biopsies
2104	35, 42, 49, 56	Liver biopsies
2112	77	Liver, kidney
2098	10	Liver, spleen, lymph gland, adrenal, myocardium, kidney

Generalised icterus was present in Nos. 2095 and 2114, being, however, of slight degree in the former. The liver was pale in animals Nos. 2095, 2097 and 2103, pinkish-yellow in colour and somewhat swollen and fatty on incision. In No. 2103 there appeared to be some irregular areas of congestion. This macroscopic appearance was quite distinct from that seen in animal No. 2114 dying with a zonal (periportal) lesion, where the liver was orange-brown in colour, more friable, and showed a distinct fine mottling on the surface or on incision. The bile was a clear, bright green.

Nos. 2103 and 2095 both had deep blackened angular erosions on the edges of the abomasal folds, together with, in No. 2103, areas of epithelial necrosis and petechiae. There was, in addition, some submucosal oedema.

Bovines Nos. 2095, 2097 and 2103 all showed a distinct pale mottling of the kidney cortex. The myocardium and endocardium of No. 2114 had irregular areas of congestion and haemorrhage.

The only finding of significance in No. 2108 was a slight orange coloration of the liver; a similar appearance was seen in No. 2116 but this animal also showed deep necrosis and erosions of the epithelium of the tip and just anterior to the dorsum of the tongue. These lesions were almost bilaterally symmetrical and closely resembled those due to *Fusiformis necrophorus* infection. It is perhaps significant that this heifer was about five months pregnant at the time of slaughter; pregnancy may have accounted for the unexpectedly severe reaction to a dose as small as 1 mg. per kg. of 150.C.47.

The carcase of Bovine 2098, which died, was pale with extensive intermuscular, periglandular and subserous, gelatinous oedema. The pharyngeal and tracheal mucosae were intensely congested and in the larynx there was extensive necrosis of the epithelium. There was an excess of clear fluid in the peritoneal and pericardial cavities; blackened ulcers were present on the abomasal folds. The liver showed small (1 mm.) white foci on the serous and cut surfaces. The associated glands showed enlargement and congestion. The kidneys were swollen and the cortex very pale, almost white; on incision the pelvis was found to be filled by gelatinous, oedematous tissue in which diffuse haemorrhages were present. The heart showed a few epicardial petechiae. There was a hypostatic congestion and oedema of the right lung.

The animal No. 2112, which had received 7·5 mg. per kg. of antrycide 77 days prior to slaughter, had lost condition, without oedema or gelatinous infiltration of the tissues on *post-mortem* examination. The kidney cortex was slightly paler than normal and showed some regular fine white streaking. The renal lymphatic glands were enlarged and haemorrhagic with some whitish foci, possibly of necrosis.

Histological Examination

Dimidium Bromide Group

Bovine 2095.—At day 35 a diffuse fatty infiltration had already commenced. At death on day 41 a few small groups of cells around the central veins were seen to be better preserved, this being especially marked in the caudate lobe.

Bovine 2114.—An extensive periportal degeneration was established by day 35. By the 42nd day pigment had begun to accumulate, especially in large vacuolated hepatic cells; it was plentiful at death on day 45. The kidney showed congestion of the boundary zone and fatty degeneration of some convoluted and collecting tubules, especially the latter.

621.C.47 Group

Bovine 2096.—A few small fat droplets were found on the 35th day in some periportal hepatic cells. By day 42 these had increased to give a well-defined, narrow zone of periportal degeneration in which the hepatic cell cytoplasm was typically pale-staining. Bile pigment was

demonstrable on days 49 and 56; fat was found only in rare hepatic cells on the latter day, but bile-duct proliferation of mild degree was quite evident.

Bovine 2110.—No abnormality attributable to the drug was detected at any time in liver biopsy material.

150.C.47 Group

Bovine 2116.—There was a centrilobular fatty degeneration of the liver accompanied by a diffuse hypertrophy of sinusoidal cells. Many of these were rounded off and apparently lying free in the sinusoids; some contained yellow-brown pigment. The sinusoids also showed increased numbers of small mononuclears and polymorphs. Since this animal was killed on day 77 when about five months pregnant it is far from certain that all the changes noted were, in fact, due to the drug.

Bovine 2097.—Died on day 33 when there was found a diffuse fatty filtration of the liver, the majority of hepatic-cell nuclei showing shrinkage and other degenerative changes. No abnormality was found in salivary gland, adrenals and spleen. The glomeruli of the kidney were congested.

Bovine 2108.—On destruction at day 40 there was evidence of slight bile-duct proliferation and of recent regenerative changes in the parenchyma. Pigment was present in some sinusoidal and portal tract macrophages. The kidney presented nothing unusual.

Bovine 2103.—The liver, on day 35, showed a diffuse fatty degeneration with some tendency to sparing of the periportal zone. At death on day 39 this change was found to be regularly distributed throughout the liver. The majority of nuclei were undergoing degeneration; polymorphs were frequent in the sinusoids.

Brownish pigment was present in fair quantity in the medullary tubules of the kidney. Many convoluted and collecting tubules contained numerous fat globules of varying size. The local muscle lesion at site of inoculation consisted of necrosis and replacement fibrosis of the fibres but cellular infiltration was not marked.

Bovine 2109.—A zonal (periportal) lesion developed in the liver of this animal, involving up to about half of the parenchyma. It was never very sharply demarcated and by day 49 there was much fat present; by day 56 no fat was left in hepatic cells. Pigment was seen in the portal tracts on days 41 and 56 p.i. with distinct proliferation of bile ducts.

Antrycide Group

Bovines 2100, 2104.—In these animals dosed at 5 mg. per kg. no changes attributable to the drug were found in liver biopsy material collected weekly between days 35 and 56 p.i.

Bovine 2112.—(7·5 mg. per kg.). Spleen and liver showed no changes attributable to the drug.

Kidney—this organ was the seat of changes probably the result of partial resolution of lesions similar to those described in Bovine 2098 (see below). It should be emphasised that Bovine 2112 was killed after 77 days whilst Bovine 2098 died 10 days after treatment.

There were streaks and more rounded foci of interstitial fibrosis and cellular infiltration through the cortex; sometimes, though by no means always, the streak-like lesions coincided with medullary rays with some extension into the outer medulla. Within these areas the tubular epithelium was low cuboidal and the basement membrane surrounded by dense collagenous strands in which numerous fibroblast nuclei were visible. Mononuclear cells and transition forms to fibro-blasts were also present, the former sometimes in dense aggregations.

The tubule lumina (particularly those of the collecting tubules) were often distended by elongated dense hyaline casts; it was noticeable that a tubule lying near the vascular pole of many glomeruli, in and near the affected areas, often showed a similar cast; this tubule appeared to be the distal limb of the loop of Henle. Smaller casts completely filled the lumina of a very large number of the narrow limbs of Henle's loops in the outer medulla. In a few instances hyaline material was present within Bowman's capsule or hyalinisation had taken place within some of the capillary loops of a glomerulus.

The convoluted tubules were usually intact; thus in a small proportion eosinophilic or hyaline material not organised into definite casts was present in the lumen.

Bovine 2098.—(10 mg. per kg.). The liver of this animal, which died on day 10 p.i., showed a centrilobular degeneration sometimes with fat globules in the affected cells. There were, in addition, numerous irregular foci of eosinophilic necrosis, usually contiguous with a central vein. Occasional single hepatic cells showed the same change. Sometimes a few polymorphs had begun to infiltrate into these foci.

Kidney—this organ showed extensive lesions which probably, to a large extent, accounted for the clinical features and gross pathological changes found in animals poisoned by this drug. Throughout the cortex the convoluted tubules were completely necrotic. The cells were swollen and granular, the tubular lumen was obliterated and no sign of nuclei remained. Glomeruli were swollen and often the capsular space was completely filled by the swollen capillary loops. The loops of Henle and the collecting tubules were relatively well preserved in that their nuclei were still clearly stained, but degenerative changes were very evident. The lumina of these were often dilated and filled by granular eosinophilic or hyaline casts. Their epithelium showed some cells filled with rounded hyaline droplets of varying size and having a shrunken pyknotic nucleus; such cells frequently became detached from the basement membrane and passed free into the lumen, either intact or after disintegration.

Accompanying these degenerative changes were cellular infiltrations composed largely of mononuclear cells, many of them large and with fairly abundant basophilic cytoplasm, also some plasma cells. Polymorphs were infrequent but formed occasional small groups within the lumen of tubules, especially in the outer medulla.

Sections of carcase lymphatic gland (prescapular) showed congestion of blood vessels and some oedematous infiltration. In the spleen the white pulp was reduced, with no lymphopoietic nodules, and macrophages with haemosiderin granules were frequent. Adrenal glands and myocardium were normal on histological examination.

Discussion

The toxic level of the three phenanthridinium compounds examined in these trials varied. It can be seen, however, that the main effects produced are similar and characterised by loss in weight and by liver damage, demonstrable histologically mainly as fatty degeneration and biochemically by elevated serum bilirubin and alkaline phosphatase.

This type of toxic damage was not demonstrable with antrycide where the main action of the drug is on the

kidney. The methods employed, therefore, were of little value in elucidating the mechanism of antrycide poisoning but it appeared that death followed a profound upset of osmotic balance between blood and tissues, probably following loss of plasma proteins through the damaged kidney tubules. The toxic effects were again delayed, but only for two to three weeks and anaemia was an added feature.

Although photosensitisation was not produced with 150.C.47, this drug seemed slightly more toxic than dimidium bromide at the same dosage levels, as judged by mortality, frequency of occurrence of weight-loss and histological and biochemical evidence of liver dysfunction. In comparison, 621.C.47 was definitely less toxic and in only one animal was there evidence of harmful effects. This was No. 2096, at 5 mg. per kg., which developed the typical delayed dimidium toxicity syndrome and transitory biochemical indications. The complete absence of reaction in the other heifer, No. 2110, at 5 mg. per kg., demonstrates the difference in susceptibility as between individuals (see also the 1 mg. per kg. dimidium bromide group). From the limited observations it seems that 3 mg. per kg. 621.C.47 was roughly equal in toxicity to 1 mg. per kg. dimidium bromide.

So far, in the work on the toxicity of phenanthridinium compounds in native cattle carried out at Kabete, the order of delayed toxicity would appear to be:—

150.C.47—dimidium bromide—621.C.47—phenidium chloride, the last showing no delayed harmful effects in all Kabete trials (Burdin *et al.*, 1952).

Summary and Conclusions

1. In a small experiment using Boran cattle, to compare the toxicity of four trypanocidal drugs, dimidium bromide, 621.C.47, 150.C.47 and antrycide, two animals were used at each of three dosage levels. The drugs were given subcutaneously and six control animals were grazed in the same paddocks.

2. Weight records were kept and weekly biochemical observations made on blood. Biopsy liver samples were examined histologically from all animals at the 5 mg. per kg. level and more extensive histological investigations carried out on necropsy material.

3. Phenanthridinium compounds all produced a syndrome of delayed liver damage associated with loss in weight, but this occurred at different dosage levels. The order of toxicity appeared to be:—

150.C.47—dimidium bromide—621.C.47.

4. Antrycide methyl sulphate, given subcutaneously at 10 mg. per kg., caused death in both animals injected, whilst 7·5 mg. per kg. of this drug caused a form of delayed toxicity differing from that with phenanthridinium compounds, in one out of two animals. The important changes found were an anaemia and nephrosis with secondary lesions dependent upon them.

Acknowledgments.—We are greatly indebted to Dr. H. S. Purchase for constant interest and advice in the course of this work and to Miss B. E. Ellis and Miss Y. L. Malherbe for considerable technical assistance. The compounds No. 621.C.47 and 150.C.47 were provided by courtesy of Dr. J. S. K. Boyd, O.B.E., M.D., D.P.H., of the Wellcome Institute for Tropical Diseases.

This paper is published by permission of Mr. R. A. Hammond, O.B.E., M.R.C.V.S., Director of Veterinary Services, Kenya.

CLINICAL COMMUNICATION

ANASTOMOSIS BETWEEN THE LARGE INTESTINE AND THE UTERUS IN A GOAT

E. A. WELLS,

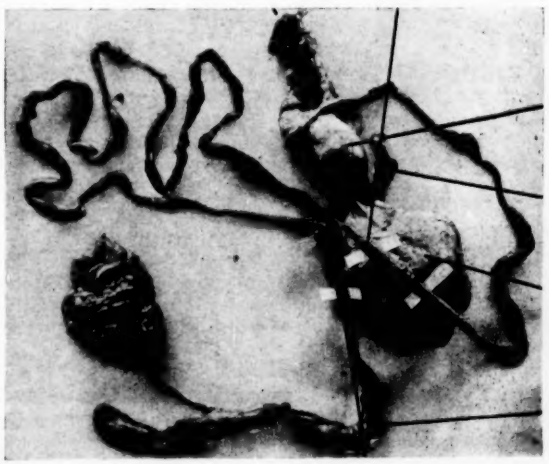
Animal Health Research Centre, Entebbe

Subject.—A black and white indigenous female goat of unknown age.

Clinical History.—On 2.7.51 the goat, heavy in kid, was seen to be anaemic and in poor condition. Two days later a large subcutaneous swelling was seen on the left chest. After a further four days (8.7.51) the condition of the animal had deteriorated, the appetite was poor, and there was a clear mucoid vaginal discharge. The temperature remained normal. A guarded diagnosis of a dead foetus was made and as the faecal worm count was found to be 1,100 eggs per gramme, the animal was treated with phenothiazine and sent back to the flock for observation.

The herdsman appeared unworried by the fact that the goat did not kid, although it continued not to thrive. The animal was found dead on 23.1.52, that is, six months after it was first seen.

Post-mortem Examination.—The abdominal cavity was found to be a mass of adhesions, the pregnant left horn being adherent to the abdominal wall. The pregnant horn contained an almost full-term kid, anteriorly presented, lying on its left side with its head detached and in the body of the uterus. The kid was in an advanced stage of decomposition and the horn contained faecal material.



A: head of foetus in body of uterus; B: non-pregnant horn; C: pregnant horn; D: caecum.

References

Burdin, M. L., & Plowright, W. (1952.) *J. comp. Path.* **62.** 178.
———, ——— & Purchase, H. S. (1952.) *Vet. Rec.* **64.** 217.
Garner, R. J. (1950.) *Ibid.* **62.** 314.
Goodwin, L. G. (1951.) Personal communication.
Plowright, W., & Burdin, M. L. (1952.) *J. comp. Path.* **62.** 189.
——, —— & Thorold, P. W. (1952.) *Ibid.* 141.

Dissection revealed anastomotic connections between both the colon and the rectum and the pregnant horn. The rectum contained normal but sparse faecal matter posterior to the anastomoses.

Measurements were as follow: length of caecum, 47 cm.; base of caecum to first anastomosis, 12 cm.; length of lumen of anastomosis into the uterine horn, 5 cm.; length of large intestine between anastomoses, 215 cm.; length of lumen of the second anastomosis into the horn, 6 cm.; second anastomosis to anus, 110 cm.

Discussion

Trouble had been experienced in this flock with castrated males butting females. Such an occurrence could have accounted for injury on July 4th, 1951. It is surmised that a rupture of the pregnant uterine horn occurred at the same time, resulting in an aseptic peritonitis which caused the adhesions. Close apposition of the colon and rectum resulted in anastomosis.

Death is presumed to have been due to metritis; but as this occurred six months after the time symptoms were first shown, and as the faecal matter in the horn was not gross, and moreover as normal but sparse faecal matter was found in the rectum, it is very probable that some intestinal contents passed through the uterus *en route* to the anus.

ANNOTATION

The Susceptibility of Man to the Virus of Foot-and-Mouth Disease

Although many cases of foot-and-mouth disease in man have been recorded in the past, reported cases have been far less frequent in recent years since the development of techniques for isolation and study of the virus. Previously, many cases of stomatitis in man, sometimes accompanied by vesicles on the hands and feet, were attributed to foot-and-mouth disease but it is very doubtful how many of these were, in fact, due to this infection. Many of the reports of alleged human infection have emanated from Germany, but McBride (1869) in this country recorded a number of cases collected from 22 different sources and he regarded these as undoubted infections of foot-and-mouth disease. Some human patients were believed to have contracted infection directly from affected cattle but the majority were thought to have become infected from milk.

Several workers have been successful in setting up foot-and-mouth disease in cattle with suspected human material. In 1922 several human cases of stomatitis characterised by the presence of vesicles occurred in Italy in the province of Como. Vesicular fluid from one patient was taken to Milan and there inoculated into a calf and guinea-pigs by Pancera (1922). All the animals developed lesions typical of foot-and-mouth disease. Rinjard, Gratiolet and Chabrot (1939) inoculated a cow with epithelium from plantar lesions in a Frenchwoman; the results were positive and the virus was identified by guinea-pig tests as the "O" type. On the other hand, Lebailly (1921) was unable to set up infection in cattle with material of human origin.

Apart from Pancera, other workers have produced lesions in the guinea-pig by inoculating human vesicular fluid and ground vesicle wall into the pad of the guinea-pig's foot. Magnusson (1939) diagnosed four cases in farm workers by this method and isolated the "O" type virus, while others have been so diagnosed by Richter (1938) and Stenström (1941).

The production of vesicles in the guinea-pig, however, is not conclusive proof of the presence of the virus of foot-and-mouth disease in the human material. Apart from the risk of accidental contamination with virus of animal origin, both herpes simplex and vaccinia are capable of producing vesicles when given by this route. Infection with the former virus is very prevalent in man and some of the clinical features attributed to human foot-and-mouth disease bear a close relationship to some of those associated with attacks of herpes simplex. On the other hand, when considering the diagnosis of human infection by the inoculation of material into guinea-pigs, it must be remembered that virus originating from cattle and pigs is not always readily infective for the guinea-pig (Foot-and-Mouth Disease Research, Interim Report, 1952), and the same could quite easily be true of virus from human lesions.

The virus has not only been isolated from suspected clinical cases of human foot-and-mouth disease but its recovery, by guinea-pig inoculation, from the faeces of a healthy farm worker who had drunk raw milk has been reported by Kling, Huss and Olin (1939). The virus thus obtained was found to be of the same type as that affecting animals on the farm.

Complement fixation tests have been made with the serum of patients believed to be convalescent from foot-and-mouth disease. Oláh and Kanyó (1938) obtained well-defined, but incomplete, fixation, with serum taken on the 20th, 32nd and 42nd days after the first observation of sickness in the patient, while Magnusson (1939) obtained positive results in sera taken from four farm workers believed to have suffered from the disease. Stenström (1941) reported in some detail a case in a milker from an affected herd. From vesicles on his hands "O" type virus was isolated by guinea-pig inoculation and during the patient's convalescence his serum showed the presence of "O" type antibodies.

Attempts to produce infection in human beings by inoculation of foot-and-mouth virus have not been successful. Lebailly (1921) failed to transmit to man virus of bovine origin by any of three different methods, and Nicolle and Balozet (1933) inoculated three persons with each of the three strains, A, O and C. With each strain one person was injected intradermally in the palm of the hand and the sole of the foot, one in the mucous membrane of the cheek and the gum, and one intramuscularly in the right thigh. No reaction, either local or general, followed these inoculations and blood taken 24, 48 and 72 hours after injection was not infective for guinea-pigs and swine. Guinea-pigs inoculated with the original material developed lesions as expected. Magnusson (1939), in two cases, attempted to transmit virus of human origin to three separate persons and failed. In addition to these deliberate inoculations of active virus into human beings, Flaum (1939) has pointed out that many thousands of persons must have been inadvertently inoculated during smallpox vaccination with calf lymph infected with foot-and-mouth virus, without any resultant human infections being reported.

One case of the disease in a laboratory worker following accidental inoculation has been reported (Pape, 1921). Pape, who later, with Waldmann, discovered the method of infecting guinea-pigs with foot-and-mouth virus, cut his thumb on a broken glass container while collecting virus material from a pig. Two days later he developed headache and shivering and on the third day vesicles appeared on the palms of his hands and the soles of his feet. During the

next two days further vesicles appeared at these sites but not elsewhere. The only buccal lesions were slight swelling and tenderness of the gums. Unfortunately, this occurrence was before the discovery of the susceptibility of guinea-pigs, and animal inoculations were not made. The clinical syndrome reported by Pape is very similar to that described in the authentic cases of the disease in man. Most of these correspond to an acute infection with an incubation period of two or three days, a preliminary phase characterised by pyrexia, headache, shivering fits, possibly tonsillitis or pharyngitis, followed by the appearance of vesicles, sometimes in the mouth but more frequently on the hands and feet. Recovery is rapid and seldom takes longer than 14 days.

The position appears to be that the susceptibility of man to the virus of foot-and-mouth disease is of an extremely low order. The great majority of alleged human infections are based upon very slender evidence and do not stand up to critical examination. However, there can be little reasonable doubt that on one or two occasions true infection of man with the virus of foot-and-mouth disease has occurred. The number of authentic cases is infinitesimal when compared with the many hundreds of thousands of persons who must have had direct contact with the virus during the period that observations have been recorded. The incidence is far too low for human infection to constitute a medical problem.

Alan O. Betts.

References

Flaum, A. (1939.) *Acta. path. microbiol. scand.* **16.** 197.
Foot-and-Mouth Disease Research, Interim Report, 1952.
Kling, C., Huss, R., & Olin, O. (1939.) *C.R. Soc. Biol., Paris.* **131.** 478.
Lebailly, C. (1921.) *C.R. Acad. Sci., Paris.* **172.** 1140.
Magnusson, H. (1939.) *Skand. VetTidskr.* **29.** 152 ; Abst. *Vet. Bull.* **10.** 587.
McBride, J. A. (1869.) *Brit. med. J.* **ii.** 536.
Nicolle, C., & Balozet, L. (1933.) *Arch. Inst. Pasteur, Tunis.* **22.** 193.
Oláh, G., & Kanyó, B. (1938.) *Állatorv. Lapok.* **61.** 260 ; Abst. *Vet. Bull.* **10.** 172.
Pancera, A. (1922.) *Clin. vet. Milano.* **45.** 251 ; quoted by Arkwright, J. A. (1928.) *Lancet.* **i.** 1191.
Pape, J. (1921.) *Berl. tierärztl. Wschr.* **37.** 354. Abst. *Rev. gén. Méd. vét.* (1921.) **30.** 650.
Richter, W. (1938.) *Arch. Derm. Berl.* **176.** 575.
Rinjard, P., Gratiolet, G., & Chabrot, J. C. (1939.) *Bull. Acad. vét. Fr.* **12.** 325.
Stenström, T. (1941.) *Acta. med. Scand.* **107.** 372.

SOIL SURVEY OF GREAT BRITAIN

Good progress in mapping the soils of Great Britain is recorded in the fourth report of the Soil Survey Research Board, and an additional 269,000 acres have been surveyed. Work has continued in Lancashire, Cambridgeshire, North Wales, Morayshire, Kincardineshire, Angus, Roxburghshire and Ayrshire and, also in Scotland, some 6,000 acres, south of the Dee between Aboyne and Banchory, have been resurveyed and the hill land mapped in more detail. The survey of a new area, mainly situated in Hertfordshire, east of Aylesbury, has begun; this will provide more information on the soils developed on the chalk and associated deposits.

The printing of soil maps of the districts around Wem, Glastonbury, Insch and Banff is well advanced and the reports are being prepared.

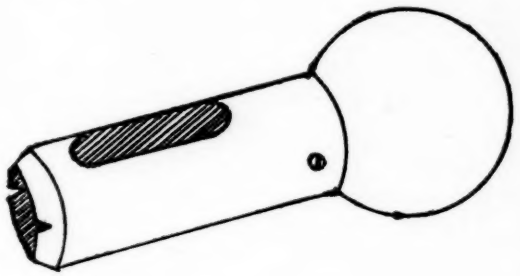
The report was published by H.M. Stationery Office on October 13th.

ABSTRACTS

A Practical Calf Dehorner*

The disadvantages of the chemical methods of disbudding calves are well known to agricultural practitioners. Actual cautery, either vulcanic or electrical, may present problems of inconvenience.

The author constructed a surgical disbudder from steel tubing with an outside diameter of 1 inch (length not stated, but 3 inches would be proportionate). The handle end is protected by a wooden ball-grip sunk into the tube and secured by a pin. The cutting end is hardened and ground to a keen edge carrying two 3/32-inch V-shape nicks. The shaft is perforated by a window 1¼ inches above the cutting edge to facilitate the clearance of removed buds which lodge.



This instrument works on the principle of the trephine. The writer normally dishorns calves from two weeks of age to three months. After cutting the peripheral skin by a circular motion the tube is tilted sideways and the isolated horn with its corium is rapidly gouged out. Haemorrhage is seldom excessive.

J. P.

*A Practical Calf Dehorner. Roberts, S. J. (1952.) *J. Amer. vet. med. Ass.* **120.** 193-195 (4 photographs.)

* * * * *

Application of the New Hypotensive (Methonium) Drugs in Veterinary Practice*

Penta- and hexamethonium compounds block impulses through the ganglia of both the sympathetic and parasympathetic nervous systems. They lower blood pressure and reduce gastric secretion. Paton (1951) considers that they reduce blood pressure only by the release of autonomic tone, and that the sympathetic tone once abolished, repeated doses have no further effect. As a result of their action, the normal pressor and buffer reflexes are inhibited and alterations of posture of the subject result in altered local blood pressure. In the prone subject, however, the new low blood pressure remains remarkably constant.

Using as a guide the dosage suggested by Lewis (1951) in human surgery, Clark and Weiss intravenously injected four nembutal-anaesthetised dogs at a standard initial dosage of 20 mg. pentamethonium iodide per 100 lb. bodyweight. Within 30 seconds of injection the mean femoral blood

* Possible Applications of the New Hypotensive (Methonium) Drugs in Veterinary Practice. Clark, R., & Weiss, R. B. (1952.) *J. S. Afr. vet. med. Ass.* **23.** 15-20.

pressure fell from 110 to 135 mm. /Hg to 65 to 90 mm. with the exception of one subject, in which repeated doses failed to lower the MFBP below 100 mm. /Hg. In general, once the desired systolic pressure of 55 mm. /Hg was reached (below which the femoral pulse was hardly perceptible), it remained at this level for 25 to 50 minutes, after which further dosage was necessary.

An initial dose of 50 mg. per 100 lb. bodyweight is suggested for the horse, but only 20 mg. for goats, and in the absence of data concerning bovines, a similar rate of initial dosage is recommended.

The advantages to the surgeon of operating under induced vascular hypotension are that incisions through muscles are practically bloodless and that seepage is reduced to a minimum. Small severed vessels dry up rapidly. Radical operations about the withers of the horse, for example, would be simplified considerably. Care is needed that the method is not employed to control haemorrhage in subjects in which the blood pressure already is dangerously low. Further, since the methonium compounds are excreted via the kidneys, they should not be used where impaired kidney function is known or suspected. On the other hand, moderate excess dosage of normal subjects appears to be attended by no toxicity. If necessary, their action can be reversed by methedrine or the strophanthine derivative K-strophanthosid (Sandoz).

The following compounds were employed :—

" Antilusin " (pentamethonium iodide) and " Hexathide " (hexamethonium iodide), both by Allen and Hanburys ; " Vegolysen " (hexamethonium bromide), May and Baker.

J. P.

REVIEW

[Incubation and Hatchery Practice. Bull. 148, Ministry of Agriculture and Fisheries. Published by H.M. Stationery Office at 3s.]

It has for long been the reviewer's belief that the veterinary profession must play a more important part in the control of poultry diseases, and that in the minds of many practitioners one of the main deterrents to undertaking this work has been a lack of knowledge of poultry practice and management. The day-old chick trade is not only a large specialised and well-organised industry, but its influence on the poultry industry as a whole is greater than that of many other sections. The hatcheries of England and Wales are responsible for the supplying of the majority of chicks purchased by farmers in this country and on their efficiency depends the economic success of the industry. They are a " bottle-neck " through which most poultry stock in the country is canalised and as such they can influence stock improvement and disease control rapidly over the whole country.

In the foreword to this Bulletin it is stated that about 100,000,000 eggs are used for hatching annually and that some 30 per cent. of this number fail to hatch—a heavy economic loss to the country. Much of this loss may be associated with nutrition, breeding or managemental factors, but disease plays a most important part and it has been said that infection with salmonella organisms is one of the greatest limiting factors to large-scale incubation.

This Bulletin is a useful guide rather than a reference book on chick production but nevertheless it outlines concisely and in logical sequence the factors influencing modern methods of chick production from the handling of hatching eggs to the dispatch of the day-old chick. Its perusal will profit veterinary practitioners and students interested in the welfare of this highly specialised branch of agriculture.

The subject is dealt with under the chapter headings of Structure of the Egg, Embryology, Selection and Handling of Hatching Eggs, Physical Requisites for Successful Incubation, Natural Hatching and Artificial Incubation, Hatchery Design, Hygiene and Management, while there are two appendices dealing with the locating and correction of incubation faults, and the hatching of turkey, goose and duck eggs.

Although egg-borne diseases are not dealt with in any detail, the chapter on hygiene is complete for most routine purposes and is based on the rules of the Ministry of Agriculture's Poultry Stock Improvement Plan. The pages devoted to the malpositions of the embryo can be of little value to the average reader since their practical significance is not fully understood, and the space could have been used to better purpose in dealing with the nutrition of the breeding flock in relation to this problem and the effects of nutritional deficiencies on hatchability and chick viability which are discussed in one short paragraph.

There are 62 pages of clearly printed text, well illustrated and supported by a list of text-books on the various subjects influencing the successful production of day-old chicks.

IN PARLIAMENT

Lords Debate Experimentation on Animals

When Parliament re-assembled on Tuesday of last week, Lord Dowding, in the Upper Chamber, drew attention to the unnecessary suffering caused by surgical and medical experiments on animals, and moved for papers. He said that people who were normally humane had the idea that the suffering inflicted on animals by surgical experiments helped to reduce the suffering of human beings. Even if it could be conclusively proved that the human being benefited directly from the suffering of animals, its infliction could be unethical and wrong. The law designed for the protection of animals against cruelty was gravely defective. There was neither adequate inspection nor proper enforcement. There was a case for an adequate enquiry into the new conditions which had arisen since the issue of the report of the Royal Commission of 1912. Means should be devised for enforcing the Act of 1876, and there should be a tighter control on the process for granting permits to experiment on animals. There should be a stop on the indefinite repetition of experiments which had already been made, and an enquiry into the vast new field of animal exploitation in the manufacture and testing of drugs.

Lord Hailey feared that the result of an enquiry would be once more to stimulate the energy of the Anti-Vivisection Society, to encourage them once more to advocate total abolition of all forms of experiment on animals, with the result that science would be deprived of the means of achieving advances in physiological science and the means of testing and standardising drugs, and the means of testing new processes as applied to medical science.

Viscount Mersey, who said that he was the Secretary to the Royal Commission on experiments on animals, was quite sure that the impression left on the mind of every member of that Commission was that the greatest care was exercised by the Home Office in supervision and adherence to the regulations.

Lord Webb-Johnson said that for eight years as President of the Royal College of Surgeons he had refused to sign many applications for experiments either because the experimenter was not working under satisfactory conditions or because the experiment had been repeated a sufficient number of times to prove that the results obtained were actual facts.

He was satisfied that the precautions taken in this country were stricter than in any other country in the world. They must not shrink from doing experiments on animals which might involve pain, and might require the survival of the animal after the operation, any more than an experimental worker when necessary did not shrink from experimenting on himself. He had put 69 stitches into himself to satisfy himself and others as to what was the most suit-

able material. Who could fail to be moved by Pasteur sucking the saliva from the mouth of a mad dog in order to make sure he had the poisonous virus? The suggestion that the general body of experimental workers were indifferent to the suffering of the animals upon whom they experimented was far from the truth.

Dramatic facts had been brought to light by these experiments. One was that the placing of an artificial valve in the great aorta, carrying the blood all over the body, had proved with benefit that an incompetent aortic valve could be replaced with a plastic valve. He had seen a dog which had been operated on eight months before and was raced around the track attached to the laboratories. The only difference he could see from a normal dog was that, instead of the normal beat, there was the click of the artificial valve. These things were worth doing. Human lives had been saved by them. They were only raising the curtain on operations on the heart itself. The heart could be stilled while an operation was performed on it. Although he sympathised with Lord Dowding's anxiety, he hoped he would not press the motion because nothing must be done to interfere with the progress of science.

Government Reply

The Earl of Selkirk, Lord in Waiting, said that the number of experiments had increased considerably since the Cruelty to Animals Act, 1876, was passed, but in practice almost all this work was carried out under certificate (a) of the Act—that was without an anaesthetic—and consisted of inoculations, applications of substances, or the administration of a drug or toxin. When the certificate was granted the inspector had to recommend that the applicant's knowledge and experience were adequate to perform an experiment and that the place was suitable. When the inspectors felt they could not give a recommendation the matter was referred to the advisory council, a panel appointed by the Secretary of State from the Royal Society, the Royal College of Physicians, the Royal College of Surgeons, and recently the Secretary of State had decided to add a person nominated by the Royal College of Veterinary Surgeons. It was clear that the whole purpose of the Act was to reduce suffering to the absolute minimum, and the question which remained was whether the inspectors were sufficiently rigid in enforcing the letter and terms of the law.

The duties of inspectors were to visit regularly all the places licensed, and this meant a visit of nearly once a quarter. In nearly all cases visits were unannounced. The chief inspector received 1,200 reports from his sub-inspectors and he presented an annual report to Parliament in which were recorded the irregularities brought to his attention. He also scrutinised all publications dealing with experiments, and satisfied himself that each had been carried out under proper authority. The chief inspector assured him that he was fully satisfied that he knew what was going on in every place on his register in the country. It was true there had been no prosecutions under the Act, but that was not a wholly satisfactory way of judging its operation. Certificates and licences had been refused, warnings had been issued, and in certain cases matters had been brought to the heads of departments or senior officers of the universities concerned.

In 1948 the Home Secretary had received a delegation who presented a number of specific cases. All these had been carefully investigated, and Mr. Ede came to the conclusion that no sufficient grounds existed either in respect of the law, or its administration to justify the appointment of a fresh Royal Commission, and there was nothing which had arisen recently to cause Sir David Maxwell Fyfe to alter that opinion.

The motion was, by leave, withdrawn.

. . . . . .

QUESTIONS

Horse Slaughter: Appointment of Committee

In the House of Commons on Monday last, Sir John Crowder asked the Minister of Food if he would state the names of the members and the terms of reference of the Committee which is to enquire into the question of the slaughter of horses.

Mr. E. Johnson asked the Minister of Food when he will be able to make a statement about the findings of the Committee set up by himself and the Secretary of State for Scotland to consider the law relating to the slaughter of horses in slaughterhouses and knackers' yards.

Major Lloyd George: I am pleased to say that the Duke of Northumberland has accepted the Chairmanship of the Committee. I will arrange for details of the membership and terms of reference to be circulated in the official report.

I hope that the Committee will hold its first meeting within the next few days but I cannot, of course, forecast when their report will be completed.

Sir J. Crowder: Could the Minister, in the meanwhile, while awaiting the report, give special instructions to his officers to do all that can be done to see that cruelty in the slaughtering of the horses is minimised as far as possible?

Major Lloyd George: As my hon. Friend probably knows, I have now been instructed as Minister to take charge of that side of slaughtering. An order was made some time ago making it incumbent on slaughterers to give notice of intention to slaughter. It is, of course, the local authority who is responsible, but I am in touch with the local authorities in this matter.

Mr. Beswick: Can the Minister say whether the terms of reference will permit the Committee to enquire into the allegations that New Forest ponies are rounded up and slaughtered for human consumption?

Major Lloyd George: I should think so, because the terms of reference refer to horses generally, but I will bring that point to the notice of the Committee.

Mr. Hastings: Do the terms of reference include the slaughtering of horses imported from abroad?

Major Lloyd George: I should have thought so because the terms of reference begin with the words: "To consider the law and the practice thereunder relating to the slaughter of horses in slaughterhouses. . . ." It does not say where they come from.

Mr. Woodburn: Will this Committee look into the cruelty that takes place in the transit of horses for slaughter?

Major Lloyd George: That is not in the terms of reference.

Mr. Rankin: Could the Minister say if the terms of reference will cover those horses which are exported to other countries and then slaughtered there?

Major Lloyd George: That would not come within the terms of reference, because it might be outside our authority altogether. If horses are exported from other countries that can only be stopped by the ordinary approach through the usual channels.

Mr. Rankin: But is it not the case that if the terms of reference do not include horses exported from this country to other countries the whole purpose of the enquiry will be rendered null and void?

Major Lloyd George: I am sorry; I misunderstood the hon. Gentleman. I will look specifically into the terms of reference with regard to his point about trade with other countries.

Following is the information.—The membership of the Committee is: The Duke of Northumberland, Chairman; Alderman L. G. H. Alldridge; The Lord Digby; Professor F. Grundy; R. Jarvis, Esq.; W. M. Mitchell, Esq., M.R.C.V.S.; Colonel Thompson.

The terms of reference are: "To consider the law and the practice thereunder relating to the slaughter of horses in slaughterhouses and knackers' yards and to recommend whether any further safeguards should be introduced to ensure the humane slaughtering of horses in this country."

NOTES AND NEWS

Diary of Events

Oct. 28th.—Meeting of the South Eastern Division, B.V.A., at Maidstone (Royal Star Hotel), 6.30 p.m.

Oct. 30th.—Joint Meeting of the Sussex Division, B.V.A., and the Brighton Division, B.M.A., at Brighton (Hotel Metropole), 8.30 p.m.

Oct. 31st.—Autumn Meeting of the Eastern Counties Division, B.V.A., in the Department of Animal Pathology, Milton Road, Cambridge, 12 noon.

B.V.A. Committee and Council Meetings

It has been decided to hold the next Committee and Council Meetings of the B.V.A. on Wednesday, Thursday and Friday, November 5th, 6th and 7th, 1952, in the following order:—

Wednesday, November 5th:

12 noon. Parliamentary and Public Relations Committee.
2.30 p.m. Veterinary State Medicine Committee.

Thursday, November 6th:

10.0 a.m. Organising Committee.
12 noon. Home Appointments Committee.
2.15 p.m. General Purposes and Finance Committee.

The above meetings will be held at 36, Gordon Square, London, W.C.1.

Friday, November 7th:

11.0 a.m. Meeting of Council at the Connaught Rooms, Great Queen Street, W.C.2.

Nov. 6th.—Meeting of the Society of Women Veterinary Surgeons, at the Royal Veterinary College, Camden Town, 2.45 p.m.

Nov. 6th.—Joint Meeting of the Central Division, B.V.A., and the Society of Women Veterinary Surgeons, at the Royal Veterinary College, Camden Town, 6 p.m.

Nov. 12th.—Meeting of the Editorial Executive Committee, B.V.A., at 36, Gordon Square, W.C.1, 2.30 p.m.

Nov. 12th.—R.S.I. London Sessional Meeting: Benjamin Ward Richardson Lecture on the hygienic and economic utilisation of offal and by-products of food animals, by H. E. Bywater, M.R.C.V.S., Chief Veterinary Officer, West Ham.

Nov. 20th and 21st.—B.V.A. Conference on Metabolic Disorders and Other Problems Related to Grassland and Fodder Crops and Innovations in Animal Husbandry, to be held in the Auditorium of the Wellcome Foundation, 183, Euston Road, London.

R.C.V.S. Examinations

Animal Management Examination.—Tour commences with London on Friday, November 7th, 1952.

Pharmacology Examination (Revised Syllabus).—Written: Friday, November 7th, 1952. Orals and Practicals: Commencing November 10th or 11th.

Membership Examinations.—Written: Thursday and Friday, December 4th and 5th, 1952. Orals and Practicals: Commencing Monday, December 8th, 1952.

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FOOT-AND-MOUTH DISEASE

In the House of Lords on Tuesday last, Lord Carrington, Parliamentary Secretary to the Ministry of Agriculture, replying to a question by the Earl of Listowel, said that in recent weeks the amount of the disease in this country had been so much reduced that after midnight that night, if all went well, there would be only one infected area. Since November 1st, 1951, there had been 582 outbreaks in all and the compensation payable to farmers amounted to about £2,738,000.

On the whole the position in western Europe was much less ominous than it was a year ago, and there was at present no reason to expect this autumn an invasion of the disease from the Continent such as we experienced last autumn and earlier this year.

The Gowers Committee which was appointed to review the policy and arrangements for dealing with the disease in Great Britain was now taking evidence, and he hoped it would be able to report in six to nine months' time.

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PERSONAL

FAO Fellowships

Professor Frank H. Manley, M.V.Sc., M.R.C.V.S., writes from Baghdad on October 11th: Dr. Mohammed Abdullah El-Dabbagh, Veterinary Inspector of the Southern Area, leaves this week to take up a Fellowship awarded by the Food and Agriculture Organisation of the United Nations. Dr. Dabbagh will study protozoology and parasitology for one year at the London School of Tropical Medicine and Hygiene and at the Parasitology Division of the Veterinary Laboratory of the Ministry of Agriculture, Weybridge, England. On his return he will serve as parasitologist in the veterinary laboratory. This is the first of four Fellowships which may be awarded by FAO to veterinary laboratory workers in Iraq.

. . . . .

Appointments.—The following appointments in the Colonial Veterinary Service are announced: Mr. S. E. Piercy, Ph.D., M.R.C.V.S., Senior Veterinary Research Officer, Kenya, to be Chief Veterinary Research Officer, Kenya; Mr. J. F. C. Swan, M.R.C.V.S., D.V.S.M., Senior Veterinary Officer, Northern Rhodesia, to be Chief Animal Husbandry Officer, Northern Rhodesia. First appointments in the Service are announced of Mr. M. C. Butler, B.Sc., M.R.C.V.S., as Veterinary Officer, Kenya, and of Mr. R. H. Johnson, B.V.Sc., M.R.C.V.S., as Veterinary Education Officer, Nigeria.

. . . .

Births.—Douglas—On October 6th, 1952, at Shrublands Nursing Home, Croydon, to Elizabeth (*née* Mennie), wife of S. W. Douglas, M.R.C.V.S., of the Royal Veterinary College, London, N.W.1, a daughter—Margaret Elizabeth.

Olivant.—On September 30th, 1952, to José and John M. Olivant, a daughter—Ann Elizabeth.

. . . . .

Marriages.—Lancaster—Alvis.—On October 11th, 1952, at St. James's Church, Fitzhead, near Taunton, Gillian Mary Alvis and Maurice Church Lancaster, B.Sc., M.R.C.V.S.

Muir—Malcolm.—On October 14th, 1952, at St. Michael's Church, Alnwick, by the Rev. Eyton Lloyd, Robert, only son of Mr. and Mrs. William Muir, Fiveways, Hurlford, to Moyra, only daughter of Mr. and Mrs. Maurice Malcolm, Park Farm, Alnwick.

. . . . .

R.A.V.C. Short Service Commission.—The following announcement is made in the issue of *The London Gazette* (*Supp.*) dated October 10th, 1952: Regular Army—Short Service Commission: Philip Taylor Williamson, B.Sc., M.R.C.V.S., to be Lieut., August 11th, 1952.

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R.C.V.S. OBITUARY

Barrett, Henry Arthur, 71, Princes Street, Nuneaton, Warwicks. Graduated Edinburgh, July 17th, 1884. Died October 10th, 1952; aged 90 years.

Sugden, Frank Haigh, Stanton House, Grantham, Lincs. Graduated London, December 17th, 1902. Died October 15th, 1952; aged 74 years.

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GENERAL OBITUARY

Mr. Philip H. Mitchiner, C.B., C.B.E., M.S., F.R.C.S.

It is with deep regret that we record the death, which occurred suddenly on Wednesday, October 15th, of Mr. Philip Henry Mitchiner, C.B., C.B.E., T.D., M.D., M.S., F.R.C.S., consulting surgeon to St. Thomas's Hospital, a former Deputy Vice-Chancellor of the University of London and Vice-President of the Royal College of Surgeons. Mr. Mitchiner, who was 64 years of age, had seen much war service and was for many years officer-in-charge of the Medical Unit of the University of London O.T.C. He had been hon. surgeon to the King since 1932.

Mr. W. G. R. Oates, Registrar of the Royal College of Veterinary Surgeons, writes: "Mr. P. H. Mitchiner was appointed as representative of the University of London on the Council of the Royal College of Veterinary Surgeons in place of Mr. J. B. Hunter, C.B.E., M.C., M.A., M.Ch., F.R.C.S., on his decease. Although Mr. Mitchiner only sat on the Council since his appointment on November 21st, 1951, in this short time he had endeared himself to his fellow members of Council by his ready wit and his humanity. As a non-veterinarian, at his specific request, he was not placed on any of the Committees of Council for he felt that his representation was confined to broad principles affecting education and the relation of the Universities with the Council. This did not prevent him, in Council, from giving excellent advice based on his experience as a member of the Council of the Royal College of Surgeons."

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ROYAL COLLEGE OF VETERINARY SURGEONS

Accessions to the Library for the Quarter Ending September, 1952

Bauer, H. R.: Ferkelverluste. Hannover, Schaper, 1950.

Boddie, G. F.: Introduction to veterinary therapeutics. Edinburgh, Oliver & Boyd, 1952.

Bossert: Les tumeurs des os. Thesis, Lyon, 1939.

Darwin, C.: The origin of species. London, John Murray, 1898.

Darwin, C.: Journal of researches into the natural history and geology of the countries visited during the voyage of H.M.S. Beagle round the world. London, Ward, Lock & Co., 1889.

Fortuna Domus: A series of lectures delivered in the University of Glasgow in commemoration of the fifth centenary of its foundation. 1952.

Gibson: The true method of dieting horses. 1721.

Harthoorn, A. M.: The therapy of shock in the dog. Utrecht, 1951. [Thesis for Doctorate in Veterinary Science, University of Utrecht.]

Hungerford, T. G.: Diseases of poultry. Sydney, Angus & Robertson [1951]. *Second edition.*

Lacroix, J. V.: Lameness of the horse. Chicago, *American Journal of Veterinary Medicine,* 1916.

Leahy, J. R., *and* Barrow, P.: Restraint of animals. Ithaca, Leahy & Barrow, 1951.

McCay, C. M.: Nutrition of the dog. *Second edition.* Ithaca. Comstock Publishing Co., 1949.

McMeekan, C. P.: Pig production, breeding and management. Christchurch, Whitcombe & Tombs [1952].

Mason, I. L.: A world dictionary of breeds, types and varieties of livestock. Slough, Commonwealth Agricultural Bureaux [1931]. *Technical Communication No.* 8.

MERCHANT, I. A.: An outline of the infectious diseases of domestic animals. Minneapolis, Burgess Publishing Co., 1951.

NEWSOM, I. E.: Sheep diseases. Baltimore, Williams Wilkins, 1952.

OLSEN. E. M.: On coliform bacteria in milk. Kobenhavn Skandinavisk Bladforlag, 1952.

SCIENTIFIC FILM ASSOCIATION: S.F.A. catalogue of medical films. London, Harvey & Blythe, 1952.

SEMPLE, A. T.: Improving the world's grasslands. Rome, FAO, 1951.

SKEAVINGTON, G.: The modern system of farriery. London, Emans, 1838.

STEEL, J. H.: A treatise on the diseases of the ox. London, Longmans Green & Co. *Second edition*. 1887.

THORNTON, H.: The inspection of food. London, Baillière, Tindall & Cox, 1952.

TRAUTMAN, A., *and* FIEBIGER, J.: Fundamentals of the histology of domestic animals, translated and revised from the eighth and ninth German edition by R. E. Habel and E. L. Biberstein. London, Baillière, Tindall & Cox, 1952.

World list of scientific periodicals. *Third edition*. London, Butterworth's scientific publications, 1952.

* * * * *

Tuberculosis (Attested Herds) Scheme, 1950

STATEMENT SHOWING POSITION IN EACH COUNTY AS AT SEPTEMBER 30TH, 1952

The figures shown in column 1 of the statement reproduced below relate to the number of cattle on June 4th, 1951. The provisional results of the June, 1952, Agricultural Returns show a reduction of 200,000 in the cattle population of Great Britain compared with June, 1951. The next quarterly statement of Attested Herds (December 31st, 1952) will show the final June, 1952, figures.

At the end of September, 1952, there were 5,250 Supervised Herds containing over 200,000 cattle. These herds need only one clear tuberculin test in order to qualify as Attested Herds.

County	Total Cattle as at 4.6.51	Number of Attested Herds as at 30.9.52	Number of Cattle in Attested Herds as at 30.9.52	Percentage of Attested Cattle to Total Cattle
Bedford	48,984	210	12,780	26·1
Berkshire	92,331	817	51,800	56·1
Buckingham	121,185	683	38,430	31·7
Cambridge	34,108	182	10,990	32·2
Isle of Ely	23,332	36	2,000	8·6
Chester	251,247	1,423	68,770	27·4
Cornwall	275,418	1,773	60,530	22·0
Isles of Scilly	516	88	516	100·0
Cumberland	226,769	3,157	144,390	63·7
Derby	191,445	776	37,540	19·6
Devon	411,624	2,960	112,610	27·4
Dorset	159,110	1,317	77,500	48·7
Durham	119,258	1,108	38,750	32·5
Essex	131,656	971	54,500	41·4
Gloucester	203,382	1,337	72,100	35·5
Hampshire	156,926	1,406	78,630	50·1
Isle of Wight	21,888	275	10,850	49·6
Hereford	139,533	1,029	40,920	29·3
Hertford	64,182	483	29,520	46·0
Huntingdon	31,605	60	4,390	13·9
Kent	127,175	826	42,180	33·2
Lancaster	281,010	2,130	82,850	29·5
Leicester	174,334	513	28,610	16·4
Lincoln (Holland)	30,853	24	1,150	3·7
Lincoln (Kesteven)	75,212	200	10,180	13·5
Lincoln (Lindsey)	173,869	344	17,840	10·3
London	—	—	—	—
Middlesex	8,210	52	2,740	33·4
Norfolk	206,585	790	58,540	28·3
Northampton	162,475	348	23,900	14·1
Soke of Peterborough	7,363			
Northumberland	202,748	680	38,770	19·1
Nottingham	116,515	376	18,980	16·3
Oxford	113,873	832	48,470	42·6
Rutland	21,485	23	960	4·5
Salop	280,257	1,338	72,240	25·8
Somerset	329,401	2,671	130,970	39·8
Stafford	240,728	807	38,270	15·9
Suffolk, East	86,629	601	29,200	33·7
Suffolk, West	43,338	282	17,740	40·9
Surrey	54,412	637	28,300	52·0
Sussex, East	110,983	833	38,880	35·0
Sussex, West	79,150	686	39,220	49·6
Warwick	156,792	637	35,770	22·8
Westmorland	97,030	1,925	78,810	81·2
Wiltshire	209,483	1,192	90,080	43·0
Worcester	105,750	459	23,570	22·3
York, E. Riding	143,974	240	11,450	8·0
York, N. Riding	256,784	1,883	65,980	25·7
York, W. Riding	350,578	2,565	93,880	26·8
ENGLAND	6,951,495	43,985	2,117,046	30·5
WALES	961,128	25,251	554,340	57·7
ENGLAND & WALES	7,912,623	69,236	2,671,386	33·8
Aberdeen	227,510	582	39,400	17·3
Angus	65,612	230	21,310	32·5
Argyll	62,947	1,428	38,900	61·7
Ayr	153,587	2,310	143,870	93·7
Banff	56,110	145	9,290	18·3
Berwick	34,902	143	9,260	26·5
Bute	11,129	352	11,129	100·0
Caithness	28,441	239	6,400	22·5
Clackmannan	4,523	42	3,030	67·0
Dumfries	105,935	1,924	94,300	89·0
Dunbarton	17,440	313	15,200	87·2
East Lothian	16,435	92	6,270	38·2
Fife	64,362	575	39,330	61·0
Inverness	52,918	1,126	16,930	32·0
Kincardine	40,169	104	8,390	20·9
Kinross	9,753	73	5,150	52·8
Kirkcudbright	75,789	1,124	69,780	92·1
Lanark	94,703	1,593	82,220	86·8
Midlothian	24,535	307	14,930	60·8
Moray	30,581	118	8,970	29·3
Nairn	8,989	53	2,990	33·3
Orkney	49,597	627	14,640	29·1
Peebles	11,676	271	10,280	88·0
Perth	91,725	547	30,780	33·5
Renfrew	30,173	551	26,720	88·5
Ross-shire	39,929	2,178	13,000	32·6
Roxburgh	35,669	232	11,140	31·2
Selkirk	6,141	107	3,090	50·3
Stirling	41,213	631	27,950	67·8
Sutherland	9,851	679	5,190	52·7
West Lothian	15,688	226	10,460	66·6
Wigtown	74,642	868	68,110	91·3
Zetland	7,453	1,805	7,453	100·0
SCOTLAND	1,600,127	21,575	875,862	54·7
GREAT BRITAIN	9,512,750	90,811	3,547,248	37·3

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

ANTHRAX:

Beds.—Hoo Farm, Mepperhall, Shefford (Oct. 10th).

Bucks.—Borough Farm, Newton Longville, Bletchley (Oct. 14th).

Ches.—Someriord Farm, Congleton (Oct. 14th); Abbey Farm, Chelford; No. 16 Holding, Ridley, Tarporley (Oct. 16th).

Dorset.—Lane End Farm, Creekmoor, Broadstone; Mount Pleasant Farm, Creekmoor Lane, Broadstone (Oct. 15th).

Essex.—The Chase, Chigwell (Oct. 14th); Bullocks Farm, Takeley, Bishop's Stortford (Oct. 16th).

Hants.—Vice Lane Farm, Bramshaw, Lyndhurst (Oct. 11th).

Kent.—Nurstead Court, Gravesend (Oct. 10th); Kent Tor, Culverstone, Meopham, Gravesend (Oct. 11th); Bottom Barn Farm, Cudham, Sevenoaks; Westfields, Addington, West Malling (Oct. 13th); Ravenscroft Farm, Cowden, Edenbridge (Oct. 14th); Sarness Farm, Waltham, Canterbury (Oct. 16th).

Lancs.—White Moss Farm, Goosnargh, Preston (Oct. 10th).

Leics.—Tooley Park Farm, Earl Shilton, Leicester (Oct. 10th).

Norfolk.—Glebe Farm, Aylmerton, Norwich (Oct. 10th).

Notts.—Farm Institute, Brackenhurst, Southwell (Oct. 13th); Diamond Farm, Edingley, Newark (Oct. 14th).

Salop.—Bromslow Farm, Lydbury North (Oct. 16th).

Somerset.—Bennell Piggeries, Chilcompton, Bath (Oct. 13th); Cooks Farm, Compton Dundon, Somerton (Oct. 15th); Buttles Farm, Churchinford, Chard (Oct. 16th).

Staffs.—Redhill Farm, Baldwin's Gate, Newcastle (Oct. 15th).

Suffolk.—Mill Farm, Warlingworth, Woodbridge; The Griffin, Brockford, Stowmarket (Oct. 14th); Gate Farm, Lawshall, Bury St. Edmunds; 36, Horsecroft Road, Bury St. Edmunds (Oct. 16th).

Surrey.—Coombes Farm, Oaks Road, Croydon (Oct. 13th); Haxted Mead Farm, Lingfield (Oct. 14th).
Sussex.—Woodsomes Farm, Lynwick Street, Rudgwick, Horsham (Oct. 14th).
Wilts.—Bellcroft Farm, Clyffe Pypard, Swindon; Church Farm, Urchfort, Devizes (Oct. 13th); Buildings at Hindon Road, Dinton, Salisbury (Oct. 16th).
Yorks.—Myrtle Farm, Hensall, Goole; Struncheon Hill Farm, Cranswick, Driffield (Oct. 13th); Firby House Farm, Bedale (Oct. 14th).

FOWL PEST:

Essex.—15, Drapers Road, Leyton (Oct. 10th); Five Oaks Lane, Chigwell Row, Chigwell (Oct. 16th).
Isle of Ely.—13, Field Baulk, March (Oct. 16th).
Norfolk.—Seamere Fields, Seamere Road, Higham, Norwich (Oct. 10th).

SWINE FEVER:

Angus.—Brathinch Farm, Stracathro, near Brechin (Oct. 15th).
Berks.—Farley Hill Farm, Farley Hill, Reading (Oct. 15th).
Ches.—51, High Street, Tarporley (Oct. 14th).
Clackmannans.—Greenhead, Alva (Oct. 16th).
Durham.—High Rockcliffe Farm, Hurworth-on-Tees, Darlington (Oct. 15th).
Essex.—Brook House Farm, Great Tey, Colchester (Oct. 13th).
Kent.—Colinette Farm, Brasted, Sevenoaks (Oct. 13th).
Leics.—Nook Farm, Southwood, Ashby-de-la-Zouch (Oct. 14th).
Lincs.—Dogdyke Bank, Lincoln (Oct. 14th).

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THOMAS BAXTER MEMORIAL PRIZE

Mr. and Mrs. Gerald Strutt, of Terling, Essex, have been awarded the Thomas Baxter Prize for 1952. This year the Committee decided that the award should be for creative work in dairy cattle breeding and improvement, and the Committee was unanimous in its decision to honour Mr. and Mrs. Strutt

"The award thus recognises one of the great achievements in the annals of British dairy cattle breeding," states the *Farmer & Stock-breeder*. "Just as the rise of the British Friesian breed has been the outstanding pedigree stock development of the last half-century, so have the Terling and Lavenham herds been the dominating force in that breed. On the milk-producing industry as a whole, Terling and Lavenham cattle have had tremendous influence. To take one example, there are 40 bulls bearing the Lavenham prefix and 41 bearing the Terling prefix among the progeny-recorded bulls in the latest National Milk Records List—far more than are contributed by any other herd of any breed. Over the years the bulls bred at Terling and Lavenham have been so numerous, and their use so widespread, as to have brought about a definite improvement in the national herd. . . . The production of milk in their own herds, and the breeding on the grand scale of cattle that made milk in other herds throughout the land, is Mr. and Mrs. Strutt's achievement. Theirs has been the skill that so largely created and directed the Terling and Lavenham herds, and the Baxter Prize award recognises the national importance of their success."

The Baxter Prize was instituted in 1949 to commemorate the services to milk producers of Sir Thomas Baxter, and is awarded for outstanding service to milk production. In addition to the Thomas Baxter Trophy the award comprises a gold medal and £100. The first winner of the award (1950) was Dr. John Hammond. In 1951 two awards were made—to Sir Thomas Dalling and Mr. James Mackintosh.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

EQUESTRIAN EVENTS AT THE XVTH OLYMPIAD

Sir,—Brigadier Peatt's report of the British Equestrian team at the XVth Olympiad, which appeared in your issue of September 27th, was extremely interesting and instructive reading; for, in addition to the composition of the teams, both men and horses and helpers, he gave a detailed description of the courses and the performances. These details must have been much appreciated by all at home, and we would not otherwise have had the good fortune to be able to read them in our own journal.

So detailed, indeed, did Brigadier Peatt make all this information that I like Mr. McGuinness, was surprised that there could be any omissions. I had hoped that if, and when, he should comment upon Mr. McGuinness's letter he would fill in the gaps and mention that Lieut.-Colonel R. C. Crowhurst—I should correct myself and give him only his discharge rank; Major Bob Crowhurst—acted as Veterinary Surgeon with the Grand Prix des Nations team. He "vetted" (Oh! that horrible slang term, but it does slip off the tongue more easily) the animals proposed to go with this team some week or so beforehand when collected at Aldershot, and flew with them on their way out and back.

We all know the care and thoroughness with which he would carry out any duty that came his way, and although we also know that he himself would be the last to wish for any "limelight" I do think, Mr. Editor, that your "Press Record" of these events should be complete.—Yours faithfully, E. BRAYLEY REYNOLDS, Newmarket. *October 14th*, 1952.

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THE DESIGNATION "GELDING"

Sir,—It is with some reluctance I join further in the correspondence on the definition of a "gelding" as I made my views clear in my letter of September 13th, but as I, with Mr. Robb, used the term "testicular tissue" I feel I must crave your indulgence once more.

Incidentally this correspondence is a pleasant relief to the interminable letters we read of the slang term "Vet." to which I never take any exception.

I do not intend to give any further explanation of "testicular tissue." Mr. Tutt asked the question in the second paragraph of his recent letter and answered it admirably in the first sentence of his following paragraph.

Regarding Mr. Robb's query on the use of the Burdizzo on the horse, in my opinion the operation cannot be complete. The anatomy of the scrotum and testes in that species renders the operation doomed only to partial success at the most. On occasion I find it difficult to sever the cord high enough, using the emasculator, in the recumbent position under anaesthesia, let alone in the standing position, particularly in young animals.

One final question to Mr. Tutt. Would he issue a certificate stating an animal to be a gelding in such a case as he describes in his initial letter of September 6th, so that the owner could use it in offering the animal for sale?—Yours faithfully, G. WARNOCK BEGG, The Lower House, Wickham, Fareham, Hants. *October 11th*, 1952.

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INCIDENCE OF "GUT-TIE"

Sir,—About six weeks ago I was called in to see a yearling cross Hereford bullock suffering from "gut-tie." The animal made a good recovery following laparotomy and severing of the fibrous band which was strangulating the bowel.

As this is the first case of "gut-tie" I have experienced in 14 years, I would like to know the incidence of the condition in other parts of the country.

The owner of this animal castrates his own calves using the method of traction.—Yours faithfully, A. NISBET, 8, Scarrowscant Lane, Haverfordwest, Pembrokeshire. *October 13th*, 1952.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange	Sheep Scab	Swine Fever
16th to 30th September, 1952	97	5	14	—	—	20
Corresponding period in—						
1951	8	—	2	—	1	81
1950	12	—	—	—	1	48
1949	16	—	42	—	—	—
1st Jan. to 30th September, 1952	657	491	171	—	1	692
Corresponding period in—						
1951	319	28	601	—	5	913
1950	277	20	102	—	21	176
1949	166	13	408	—	33	5

Tuberculosis (Attested Herds) Schemes

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at September 30th, 1952, was as follows:—

England	Wales	Scotland	Total (Great Britain)
43,985	25,251	21,575	90,811